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Common cutaneous manifestations in hematological malignancies

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ABSTRACT

Hematologic malignancies (HMs) are among the most prevalent and diverse types of cancer. Patients with HMs may experience a wide range of skin-related issues. These dermatological manifestations can be grouped into three main categories: Direct skin infiltration by malignant cells or their byproducts, cutaneous effects indirectly linked to the malignancy, and skin changes resulting from treatment. Such dermatological issues can significantly impact a patient's quality of life. Managing these complex conditions requires a collaborative, multidisciplinary approach. Close coordination between dermatologists and hematologists is crucial for accurate diagnosis, personalized treatment planning, and ongoing monitoring.

Keywords: Hematological malignancy, Cutaneous manifestation, Skin infiltration, Pre-malignant

INTRODUCTION

Hematologic malignancies (HMs) represent one of the most common and highly heterogeneous groups of neoplastic disorders. The global burden of HMs has increased significantly, with incidence rates rising 2–3 times between 1990 and 2019. In 2019, the number of new cases of leukemia, multiple myeloma (MM), non-Hodgkin lymphoma (NHL), and Hodgkin lymphoma (HL) reached 643.58 thousand, 155.69 thousand, 457.08 thousand, and 87.51 thousand, respectively, while the number of related deaths rose to 334.59 thousand, 113.47 thousand, 254.61 thousand, and 27.55 thousand, respectively.[1]

Patients with HMs can experience a wide range of dermatological manifestations [Table 1], which can significantly impair their quality of life and, in rare cases, increase the risk of mortality.[2,3] Dermatological manifestations in HMs can be classified into three broad categories: Direct infiltration of the skin by malignant cells or their products, such as paraproteins. This includes conditions such as *Leukemia Cutis* (LC), where myeloid or lymphoid neoplastic leukocytes infiltrate the skin, leading to clinically identifiable cutaneous lesions. Other forms of cellular infiltration include lymphomatous infiltrate and plasma cell infiltrate. Cutaneous manifestations are indirectly caused by the underlying malignancy, such as paraneoplastic conditions or dermatological syndromes. Dermatological changes caused by treatments, including those related to anti-neoplastic drugs, immune-mediated diseases post-bone marrow transplant, and skin infections resulting from immunosuppression.[4]

This article focuses on specific skin lesions secondary to systemic HM infiltration, and will not address primary cutaneous lymphomas. In addition, dermatological changes induced by anti-

Table 1: Skin Manifestations of Hematological malignancies

Type/ Classification of Skin Disorders	Skin disorders	Cutaneous Features	Associated Hematological Malignancies
Cutaneous infiltration with malignant cells	Leukemia Cutis	Single or multiple, monomorphic, dark-red or hemorrhagic skin nodules-leg arms face. May present as papules, maculopapular rash or diffuse infiltration	AML (monocytic or myelomonocytic), CML, Chronic myelomonocytic leukemia (CMML)(in transformation), precursor B-cell acute lymphoblastic leukemia, CLL, and hairy cell leukemia, T-cell leukemia/lymphomas like precursor T-cell acute lymphoblastic leukemia, adult T-cell leukemia/ lymphoma (ATLL), and T-cell prolymphocytic leukemia Myelodysplastic syndrome, Myeloproliferative Neoplasms
	Lymphomatous infiltration	Polymorphous, red papules, nodules, plaques, ulcer and erythrodermic states. Occurs in the head, neck, axillary, and inguinal folds, correlating with lymph node drainage areas.	NHL subtypes-Mantle cell lymphoma, Follicular lymphoma, Intravascular lymphoma, and Lymphomatoid granulomatosis, Hodgkin lymphoma (HL)
	Plasma cell infiltrates	Epistaxis, oral bleeding, purpura, tense bullae, necrotic ulcers, and gangrene. Cutaneous macroglobulinosis appears as translucent, flesh-colored papules on extensor sites	Multiple myeloma (MM) Waldenström macroglobulinemia (WM)
Deposition Diseases	AL Amyloidosis Type 1 Cryoglobulinemia		MGUS Multiple Myeloma, Waldenstrom Macroglobulinemia
Paraneoplastic conditions (Indirect cutaneous Manifestations of Hematological Malignancies)	Sweet syndrome Pyoderma gangrenosum Subcorneal Pustular Dermatosis (SPD) Neutrophilic eccrine hidradenitis (NEH) Erythema elevatum diutinum Eosinophilic Dermatosis of Hematologic Malignancy (EDHM) Paraneoplastic pemphigus (PNP) Scleromyxedema, Scleredema Paraneoplastic Pruritus Paraneoplastic Cutaneous small-vessel vasculitis (CSVV)		AML, chronic myeloid neoplasms, and AMML, Monoclonal gammopathies, MM, or lymphoid neoplasms CLL(, chronic lymphocytic leukemia), CML chronic myeloid leukemia, IgG cryoglobulinemia AML, B-cell CLL, Chronic myelomonocytic leukemia, Hodgkin's lymphoma, and non-Hodgkin's lymphoma. IgA monoclonal gammopathy, myelodysplasia, myeloproliferative disorders, paraproteinemia, hairy cell leukemia. CLL, acute monocytic leukemia, acute lymphoblastic leukemia, myelofibrosis, chronic myelogenous leukemia, large cell lymphoma, mantle cell lymphoma, MALT lymphoma, diffuse large B-cell lymphoma. NHL, CLL, Castleman's disease, thymoma, Waldenström macroglobulinemia, HL, monoclonal gammopathy. MGUS, multiple myeloma, or plasmacytomas. Hodgkin's disease and Non-Hodgkin's disease. Myelodysplastic syndrome (MDS), AML, CML, myelofibrosis, polycythemia vera (PV), and essential thrombocythemia.
Dermatological syndromes associated with Haematological Malignancies	Schnitzler's Syndrome Polyneuropathy, organomegaly, endocrinopathy, M-protein and skin changes (POEMS) Syndrome	Urticarial rash, fevers, arthralgias, myalgias, IgM monoclonal gammopathy. Polyradiculoneuropathy, clonal plasma cell disorder, sclerotic bone lesions, elevated vascular EGF, and Castleman's disease.	Monoclonal paraproteinemia IgM and IgG Monoclonal plasma cell disorder

neoplastic drugs, immune-mediated diseases after bone marrow transplants, or infections due to immunosuppression are beyond the scope of this discussion.

INFILTRATION OF SKIN DUE TO MALIGNANT CELLS

LC

LC refers to the infiltration of the skin by neoplastic leukocytes in patients with peripheral leukemia. Non-specific cutaneous lesions occur in 30–40% of leukemia cases.[5] Most skin lesions develop in patients already diagnosed with leukemia (55–77%), but cutaneous lesions may also present at the onset of systemic leukemia (23–44%) or precede its development in peripheral blood or bone marrow in 2–3% of cases. This latter condition, termed "aleukemic cutis," often progresses to acute myeloid leukemia (AML).[6]

Any leukemia subtype can involve the skin, leading to LC, though it is most common in children with congenital leukemia (25–30%) and chronic lymphocytic leukemia (CLL) or AML with monocytic or myelomonocytic morphology. These subtypes account for 13% and 8% of LC cases, respectively.[7] Cutaneous involvement in chronic myeloid leukemia (CML) is rare and often signals the blast phase.[7]

Myeloid or monocytic leukemia associated with LC includes AML (monocytic or myelomonocytic), CML, chronic myelomonocytic leukemia (CMML) (in transformation), and myelodysplastic syndrome (MDS) (in transformation). Lymphoproliferative disorders causing LC involve B-cell leukemia/lymphomas such as precursor B-cell acute lymphoblastic leukemia, CLL, and hairy cell leukemia, as well as T-cell leukemia/lymphomas such as precursor T-cell acute lymphoblastic leukemia, adult T-cell leukemia/lymphoma, and T-cell prolymphocytic leukemia.[7] Cutaneous involvement often indicates advanced disease and warrants investigation for extramedullary spread. Occasionally, LC marks a relapse of CML.[8]

LC typically presents as nodular lesions, tumors, plaques, or erythematous, dome-shaped, rubbery, firm nodules and papules. Nodules and papules are usually firm, dome-shaped, and erythematous. Multiple nodules and papules are common in patients with myeloid LC.[5] LC rarely involves the palms, soles, or oral mucosa. The morphology and distribution of individual lesions do not specifically correlate with leukemia subtypes, though acute leukemias often present with rapidly disseminated lesions, while chronic forms show gradual, stepwise progression.[7] The clinical and morphological presentation of LC varies widely. Lesions can be localized or widespread and appear as solitary or grouped manifestations anywhere on the skin.[9] LC may also present as diffuse purpura and is a known cause of blueberry muffin syndrome in infants. Common features include papules and nodules (60%), which are typically firm or rubbery. Plaques are rare, but transitional morphologies are possible. Additional findings include erythema, erythroderma, ulcers, and blisters. Lesion colors range from red, brown, yellow, and blue to gray and purpuric. Deep lesions may appear skin colored. There is no site predilection; the head, trunk, and extremities are equally affected. Lesions may be singular, grouped, disseminated, or exanthematous. In oral mucosa, LC may manifest as gingival hyperplasia, nodules, or ulcers.[9]

Lymphomatous skin involvement

Skin involvement is more common in NHL than in HL. Common NHL subtypes include mantle cell lymphoma, follicular lymphoma, intravascular lymphoma, and lymphomatoid granulomatosis. Cutaneous NHLs are classified as T-NHL or B-NHL based on their cell origin.[10] Primary T-NHLs are more common in the skin, whereas B-NHLs primarily involve lymph nodes.[10,11] The incidence of specific lesions in NHL ranges from 13.9% to 19%, characterized by neoplastic elements in the skin.[11] T/NK secondary cutaneous lymphomas (SCLs) frequently affect the extremities, whereas B-cell lineage SCLs primarily involve the trunk.[5] In HL, skin involvement often occurs in the head, neck, axillary, and inguinal folds, correlating with lymph node drainage areas. Direct extension from an underlying lymph node can also occur.[12]

Clinically, skin lesions in lymphomas are polymorphous, presenting as red papules, nodules, and plaques, often with ulcerations and erythrodermic states.[13] Skin lesions indicate disease progression and poorer prognosis due to aggressive disease behavior.[14,15] Specific manifestations are less frequent than non-specific ones, as the skin is rarely an early site of metastasis but often a late-stage target due to systemic neoplastic cell circulation.[15]

Plasma cell malignancies

MM and Waldenström macroglobulinemia (WM) are the most common plasma cell malignancies. In addition to bone marrow, neoplastic plasma cells can develop in extramedullary sites, such as solitary plasmacytomas, most often in the bone and upper respiratory tract, with rare skin involvement.[4] Skin involvement in MM is extremely rare, with a prevalence of 1.9%.[16] Extramedullary soft-tissue plasmacytomas can involve the skin through direct extension from skeletal lesions or through hematogenous or lymphatic spread. Skin plasmacytomas appear as reddish, violaceous, non-tender dermal or subcutaneous nodules, occasionally presenting as a diffuse erythematous rash.[1] Biopsy typically shows infiltration by clonal plasma cells. Cutaneous plasmacytomas often arise as late complications in heavily

treated MM cases and are associated with high tumor burden, plasma cell leukemia, high-risk features, and poor prognosis.[16,17]

Cutaneous Manifestations of Plasma Cell Disorders- Accumulation of immunoglobulins (Igs) from neoplastic plasma cells in conditions such as monoclonal gammopathy of undetermined significance (monoclonal gammopathies of unknown significance [MGUS], MM, or WM manifests as macroglobulinemia cutis, primary Ig light chain amyloidosis (AL amyloidosis), and type I cryoglobulinemia.[4] Macroglobulinemia cutis results from IgM deposits, presenting as IgM storage papules or immunobullous macroglobulinemia cutis.[3] Skin manifestations include epistaxis, oral bleeding, purpura, tense bullae, necrotic ulcers, and gangrene. Typically, cutaneous macroglobulinosis appears as translucent, flesh-colored papules on extensor sites.[18] However, other studies report purplish plaques (83%), papules (25%), and tumors (42%), commonly affecting the face (50%), lower limbs (42%), upper limbs (25%), and trunk (25%), with lesions often symmetrical and bilateral.[19]

In AL amyloidosis, λ light chains predominate over κ light chains (3:1).[20] Approximately 30–40% of patients exhibit skin manifestations, including a waxy appearance, "pinch purpura," and spontaneous bleeding. Hemorrhagic lesions such as ecchymoses and purpura are common around the eyes ("raccoon eyes"). Yellow, waxy plaques or papules can appear in the periorbital area or elsewhere.[21] Bullous lesions are common in MM-associated AL amyloidosis and are often hemorrhagic.[18]

Type I Cryoglobulinemia – This condition, characterized by a single monoclonal Ig (commonly IgM, less often IgG or IgA), is associated with B-cell malignancies such as WM, myeloma, and MGUS. Cryoprecipitate formation leads to small-vessel obstruction in the skin and other tissues.[22] Clinical features include purpura (80%), Raynaud's phenomenon (25–40%), distal necrosis (30–35%), cold urticaria (90–100%), livedo (10–15%), inflammatory macules, or papules.[4,22] Vascular purpura often begins on the lower limbs but may extend to the abdomen.[22]

Cutaneous manifestations indirectly caused by the underlying malignancy

This category includes paraneoplastic conditions or dermatological syndromes.

Paraneoplastic dermatosis refers to a group of skin diseases strongly associated with internal malignancies. These conditions may precede, accompany, or occur after the diagnosis of cancer, without the presence of cancer cells in the skin.[1,2] Though not fully understood, a common factor appears to be dysregulation of the innate immune system with aberrant activation of neutrophilic granulocytes. Overexpression of pro-inflammatory cytokines such as tumor necrosis factor-α and interleukin (IL)-1 superfamily IL-1 and IL-36 is commonly observed, alongside elevated serum granulocyte colony-stimulating factor concentrations, which may also contribute to these conditions.[23]

Sweet syndrome, also known as acute febrile neutrophilic dermatosis, is clinically characterized by painful erythematous papules and nodules topped with pseudovesicles, which may merge into plaques. Systemic inflammation signs often include fever, joint and muscle pain, fatigue, and headache. Classic Sweet syndrome is diagnosed when both major criteria (sudden painful skin lesions and dense dermal neutrophilic infiltration without leukocytoclastic vasculitis) and at least two of the four minor criteria (fever; prior infection/vaccination, neoplasia, autoimmune disease or pregnancy; good response to prednisolone; and elevated inflammatory markers) are present.[23]

Sweet syndrome often affects women in their 30–60s and is typically idiopathic but can be associated with pregnancy, autoimmune diseases, or infections. It may also result from drug use or malignancy.[24] The paraneoplastic variant accounts for approximately 20% of cases, most commonly linked to HMs.[4] Hematologic associations frequently include AML, chronic myeloid neoplasms, and AMML, with rare connections to monoclonal gammopathies, MM, or lymphoid neoplasms.[25] Malignancy-associated sweet syndrome tends to involve extracutaneous sites more frequently than idiopathic forms.[26] Mucosal lesions, neurological complications (e.g., Parkinsonism), and ophthalmologic conditions (e.g., optic neuritis and chorioretinitis) are more prevalent in patients with myeloproliferative diseases. Other clinical variants include acute necrotizing, isomorphic, subcutaneous forms, and neutrophilic dermatoses of the dorsal hands. Acute necrotizing variety features a rapid onset of edematous erythematous plaques with accompanying skin and soft tissue necrosis. Isomorphic Sweet Syndrome develops at trauma sites such as radiation fields or surgical scars. Subcutaneous Sweet Syndrome overlaps with pyoderma gangrenosum, commonly associated with myeloproliferative disease, and shows a predilection for the buttocks and lower extremities without tissue necrosis.[4]

Pyoderma gangrenosum, a neutrophilic dermatosis, begins as a painful papule or pustule, progresses to a nodule, and eventually ulcerates. It is classified into four clinical types: classical ulcerative, bullous, pustular, and vegetative. Between 20% and 60% of pyoderma gangrenosum cases are associated with HMs, with approximately 70% of bullous cases linked to conditions such as myelogenous leukemia, lymphoma, monoclonal gammopathy, and MDS.[27,28]

Subcorneal pustular dermatosis (SPD) is a relapsing, symmetrical eruption favoring the trunk, intertriginous

areas (particularly the axillae), and flexural extremities. It has been associated with numerous hematologic disorders, including aplastic anemia, lymphomas, MM, CLL, CML, IgG cryoglobulinemia, and monoclonal gammopathies.[29] In cases of monoclonal gammopathies and MM, the most commonly associated paraprotein is IgA, even though most myeloma patients typically have elevated IgG M-proteins. The interval between SPD onset and myeloma diagnosis varies, with the gammopathy occasionally appearing years later.[30] Clinically, SPD presents abruptly with or without pain, sometimes pruritic superficial papules, which develop into flaccid pustules, vesicles, or occasionally bullae. Lesions spread within a day or two to form annular, circinate, or serpiginous patterns with a central clearing of peripheral pustules. It arises on normal-appearing skin or erythematous or inflamed skin. Pustules have purulent material in the bottom half and the top half containing clear fluid, known as a hypopyon pustule.[30]

Neutrophilic eccrine hidradenitis (NEH) is a rare neutrophilic dermatosis presenting as infiltrated or edematous Sweet syndrome-like papules or plaques. Histologically, it is characterized by aseptic neutrophilic infiltrates localized around eccrine coils and glands. Lesions are typically asymptomatic but may be pruritic or painful, appearing on the trunk, face, or extremities.[31] Fever is frequently observed and is often associated with neutropenia.[31] Evidence of underlying HM is present in 90% of NEH cases, with AML being the most common diagnosis. Other malignancies include B-cell chronic lymphatic leukemia, CMML, HL, and NHL. Most patients (84%) have undergone chemotherapy, particularly cytarabine and anthracyclines, before NEH onset. NEH generally resolves spontaneously within days to weeks and does not require specific treatment.[4,31]

Erythema elevatum diutinum is a rare, chronic dermatosis characterized by red-violet to red-brown papules, plaques, and nodules, typically affecting extensor surfaces. While lesions are often asymptomatic, some patients experience pain, burning sensations, arthralgia, or fever. Spontaneous resolution commonly occurs within 5–10 years. Though benign, erythema elevatum diutinum is associated with infections, hematologic abnormalities, autoimmune diseases, and systemic conditions. It frequently coexists with disorders such as IgA monoclonal gammopathy, myelodysplasia, myeloproliferative disorders, paraproteinemia, and hairy cell leukemia.[32]

Eosinophilic Dermatosis of HM (EDHM) is a chronic, relapsing, pruritic skin disorder occurring in patients with various hematologic neoplasms. It is most commonly linked to CLL but has also been observed in acute monocytic leukemia, acute lymphoblastic leukemia, myelofibrosis, chronic myelogenous leukemia, large cell lymphoma, mantle cell lymphoma, mucosa-associated lymphoid tissue (MALT) lymphoma, diffuse large B-cell lymphoma, follicular lymphoma, small lymphocytic lymphoma, lymphoplasmacytic lymphoma, marginal zone lymphoma, aggressive T-cell lymphoma, and MM/monoclonal gammopathy of undetermined significance. EDHM presents with a broad spectrum of clinical manifestations, including erythematous papules or nodules, blisters and vesicles, urticarial or cellulitis-like plaques, and cutaneous ulcerations.[33]

Autoimmune blistering dermatoses - includes paraneoplastic pemphigus (PNP), a rare and devastating autoimmune disorder affecting the skin and mucosa, strongly associated with malignancies. HMs account for approximately 84% of all PNP cases, including NHL (38.6%), CLL (18.4%), Castleman's disease (18.4%), thymoma (5.5%), WM (1.2%), HL (0.6%), and monoclonal gammopathy (0.6%).[4] Non-hematological neoplasms associated with PNP include carcinomas (8.6%), sarcomas (6.2%), and melanoma (0.6%).[2,34] In children and adolescents, PNP is most often associated with Castleman's disease and can frequently be its presenting symptom.[34] The pathogenesis involves immune system interactions with concomitant neoplasms, where autoantibodies target desmosomal and hemidesmosomal antigens, including periplakins and envoplakins.[34] The lesions are polymorphic, presenting as blisters, erosions, macules, papules, and plaques. The Nikolsky sign may be positive. Cutaneous lesions often follow mucosal involvement and commonly affect the upper body. The manifestations can mimic pemphigus, bullous pemphigoid, erythema multiforme, graft-versus-host disease, or lichen planus. Treatment outcomes remain poor, making it critical to identify and manage the associated malignancy.[34]

Scleromyxedema is a generalized cutaneous mucinosis presenting as widespread, firm, waxy, translucent papules and sclerodermatous eruptions. These lesions commonly affect the face, ears, upper trunk, arms, thighs, and dorsum of the hands. In the absence of thyroid disease, scleromyxedema frequently occurs in patients with monoclonal gammopathies, such as MGUS, MM, or plasmacytomas.[35] Scleredema manifests as ill-defined, woody, non-pitting indurated plaques, predominantly on the upper body, including the face, neck, trunk, and extremities. Plasma cell dyscrasias, particularly MGUS and MM, are commonly associated. IgG monoclonal gammopathy is most frequent, followed by the IgA type. On average, gammopathies are diagnosed 10 years after the onset of scleredema skin changes.[36,37]

Chronic prurigo is a paraneoplastic pruritus, marked by intense pruritus, characterized by symmetrically distributed papules, hyperkeratotic or excoriated nodules, and scars, particularly on the lower limbs, trunk, and other easily scratched areas. Chronic pruritus, defined as itch persisting for more than 6 weeks, has been linked to internal malignancies, especially lymphoproliferative diseases. Reports indicate a pruritus prevalence of up to 30% in

Hodgkin's disease and approximately 15% in non-Hodgkin's disease.[38]

Cutaneous small vessel vasculitis can have numerous etiologies, with HMs accounting for 90% of cases and solid organ malignancies for the remaining 10%.[39] Commonly associated hematologic disorders include MDS, AML, CML, myelofibrosis, polycythemia vera, and essential thrombocythemia.

Patients with paraneoplastic vasculitis tend to be older and lack typical precipitating factors such as infections. The pathogenesis involves abnormal clearance of immune complexes, binding of non-specific antibodies to vascular walls, and dysregulated Ig production. In hematologic cases, increased blood viscosity may impair immune complex clearance in dermal small vessels.[25]

Syndromes involving skin associated with HMs

Schnitzler's Syndrome – Schnitzler's syndrome is a rare disorder characterized by intermittent, non-pruritic urticarial rash, fevers, arthralgias, myalgias, and monoclonal gammopathy, most commonly of the IgM subtype. It is driven by IL-1 overproduction.[40]

Polyneuropathy, organomegaly, endocrinopathy, M-protein and skin changes (POEMS) syndrome

POEMS syndrome is a life-threatening paraneoplastic condition arising from plasma cell neoplasms. Diagnostic criteria include major features such as polyradiculoneuropathy, clonal plasma cell disorder, sclerotic bone lesions, elevated vascular endothelial growth factor, and Castleman's disease. Minor features include organomegaly, endocrinopathy, characteristic skin changes, papilledema, extravascular volume overload, and thrombocytosis.[41]

CONCLUSION

HMs represent a heterogeneous group of neoplastic disorders with a wide spectrum of dermatological manifestations. These manifestations can significantly affect patients' quality of life and, in some cases, increase the risk of mortality. Skin involvement in HMs may be specific, such as direct infiltration by malignant cells, or non-specific, including paraneoplastic dermatoses or other common hematological alterations such as pallor and ecchymosis.

Many of these manifestations are linked to profound immune system dysregulation, which contributes to the high prevalence of autoimmune complications observed in these patients. Additional cutaneous changes may result from treatment-related side effects or opportunistic infections.

A comprehensive dermatological examination and a thorough understanding of these skin changes are crucial. Such manifestations may represent neoplastic infiltration, signal a systemic condition, or even precede the diagnosis of an underlying HM.

Managing these complex conditions necessitates a multidisciplinary approach. Collaboration between dermatologists and hematologists is essential to accurately diagnose skin conditions, plan and tailor treatment strategies, and ensure continuous follow-up. Ultimately, this integrated care approach aims to optimize patient outcomes and provide the highest quality of care.

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